```
     Set   Items   Description
      ---   -----   -----------
? s forkhead
      S1    2842   FORKHEAD
?
<------User Break------>
!


? s Ring
      S2  556248   RING
? s ring(5n)finger
          556248   RING
          125175   FINGER
      S3    5697   RING(5N)FINGER
? s s1 and s3
            2842   S1
            5697   S3
      S4      16   S1 AND S3
? s metaphase
      S5   28366   METAPHASE
? s s4 and s5
              16   S4
           28366   S5
      S6       3   S4 AND S5
? rd
>>>Duplicate detection is not supported for File 340.

>>>Records from unsupported files will be retained in the RD set.
...completed examining records
      S7       1   RD (unique items)
? t s7/3,k,ab/1

 7/3,K,AB/1     (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2004 The Dialog Corp. All rts. reserv.

12422760   PMID: 12810945
  Epigenetic inactivation of CHFR in human tumors.
  Toyota Minoru; Sasaki Yasushi; Satoh Ayumi; Ogi Kazuhiro; Kikuchi
Takefumi; Suzuki Hiromu; Mita Hiroaki; Tanaka Nobuyuki; Itoh Fumio; Issa
Jean-Pierre J; Jair Kam-Wing; Schuebel Kornel E; Imai Kohzoh; Tokino
Takashi
  Department of Molecular Biology, Cancer Research Institute, Sapporo
Medical University, Sapporo 060-8556, Japan.
  Proceedings of the National Academy of Sciences of the United States of
America (United States) Jun 24 2003, 100 (13) p7818-23, ISSN
0027-8424   Journal Code: 7505876
  Document type: Journal Article
  Languages: ENGLISH
  Main Citation Owner: NLM
  Record type: Completed
  Cell-cycle checkpoints controlling the orderly progression through
mitosis are frequently disrupted in human cancers. One such checkpoint,
entry into metaphase , is regulated by the CHFR gene encoding a
protein possessing forkhead-associated and RING finger
domains as well as ubiquitin-ligase activity. Although defects in this
checkpoint have been described, the molecular basis and prevalence of CHFR
inactivation in human tumors are still not fully understood. To address
this question, we analyzed the pattern of CHFR expression in a number of
human cancer cell lines and primary tumors. We found CpG
methylation-dependent silencing of CHFR expression in 45% of cancer cell
lines, 40% of primary colorectal cancers, 53% of colorectal adenomas, and
30% of primary head and neck cancers. Expression of CHFR was precisely
```

correlated with both CpG methylation and deacetylation of histones H3 and H4 in the CpG-rich regulatory region. Moreover, CpG methylation and thus silencing of CHFR depended on the activities of two DNA methyltransferases, DNMT1 and DNMT3b, as their genetic inactivation restored CHFR expression. Finally, cells with CHFR methylation had an intrinsically high mitotic index when treated with microtubule inhibitor. This means that cells in which CHFR was epigenetically inactivated constitute loss-of-function alleles for mitotic checkpoint control. Taken together, these findings shed light on a pathway by which mitotic checkpoint is bypassed in cancer cells and suggest that inactivation of checkpoint genes is much more widespread than previously suspected.

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?

```
? ds

Set     Items   Description
S1       2842   FORKHEAD
S2     556248   RING
S3       5697   RING(5N)FINGER
S4         16   S1 AND S3
S5      28366   METAPHASE
S6          3   S4 AND S5
S7          1   RD (unique items)
? s s1 and s2
           2842  S1
         556248  S2
      S8      27  S1 AND S2
? s s8 and s5
             27  S8
          28366  S5
      S9       3  S8 AND S5
? rd
>>>Duplicate detection is not supported for File 340.

>>>Records from unsupported files will be retained in the RD set.
...completed examining records
     S10       1  RD (unique items)
? t s10/3,k,ab/1

 10/3,K,AB/1     (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2004 The Dialog Corp. All rts. reserv.

12422760   PMID: 12810945
  Epigenetic inactivation of CHFR in human tumors.
  Toyota Minoru; Sasaki Yasushi; Satoh Ayumi; Ogi Kazuhiro; Kikuchi
Takefumi; Suzuki Hiromu; Mita Hiroaki; Tanaka Nobuyuki; Itoh Fumio; Issa
Jean-Pierre J; Jair Kam-Wing; Schuebel Kornel E; Imai Kohzoh; Tokino
Takashi
  Department of Molecular Biology, Cancer Research Institute, Sapporo
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  Proceedings of the National Academy of Sciences of the United States of
America (United States) Jun 24 2003, 100 (13) p7818-23, ISSN
0027-8424   Journal Code: 7505876
  Document type: Journal Article
  Languages: ENGLISH
  Main Citation Owner: NLM
  Record type: Completed
  Cell-cycle checkpoints controlling the orderly progression through
mitosis are frequently disrupted in human cancers. One such checkpoint,
entry into metaphase , is regulated by the CHFR gene encoding a
protein possessing forkhead-associated and RING finger domains
as well as ubiquitin-ligase activity. Although defects in this checkpoint
have been described, the molecular basis and prevalence of CHFR
inactivation in human tumors are still not fully understood. To address
this question, we analyzed the pattern of CHFR expression in a number of
```

```
SYSTEM:OS  - DIALOG OneSearch
  File 155:MEDLINE(R) 1966-2004/May W2
        (c) format only 2004 The Dialog Corp.
*File 155: Medline has been reloaded. Accession numbers
have changed. Please see HELP NEWS 154 for details.
  File  55:Biosis Previews(R) 1993-2004/May W2
        (c) 2004 BIOSIS
  File  34:SciSearch(R) Cited Ref Sci 1990-2004/May W2
        (c) 2004 Inst for Sci Info
  File 434:SciSearch(R) Cited Ref Sci 1974-1989/Dec
        (c) 1998 Inst for Sci Info
  File 340:CLAIMS(R)/US Patent 1950-04/May 13
        (c) 2004 IFI/CLAIMS(R)
*File 340: Annual reload and classification updates delayed due
 to Dialog processing issues.

      Set  Items  Description
      ---  -----  -----------
? s (mitotic(5n)checkpoint) or chfr
           77734  MITOTIC
           14930  CHECKPOINT
            1894  MITOTIC(5N)CHECKPOINT
              68  CHFR
      S1    1921  (MITOTIC(5N)CHECKPOINT) OR CHFR
? s forkhead
      S2    2842  FORKHEAD
? s s1 and s2
            1921  S1
            2842  S2
      S3      12  S1 AND S2
? s ring(5n)finger
          556248  RING
          125175  FINGER
      S4    5697  RING(5N)FINGER
? s s3 and s4
              12  S3
            5697  S4
      S5       7  S3 AND S4
? rd
>>>Duplicate detection is not supported for File 340.

>>>Records from unsupported files will be retained in the RD set.
...completed examining records
      S6       3  RD (unique items)
? t s6/3,k,ab/1-3

 6/3,K,AB/1     (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2004 The Dialog Corp. All rts. reserv.

16146132   PMID: 15138487
   Promoter hypermethylation of the Chfr gene in neoplastic and
non-neoplastic gastric epithelia.
  Honda T; Tamura G; Waki T; Kawata S; Nishizuka S; Motoyama T
  [1] 1Department of Pathology, Yamagata University School of Medicine,
2-2-2 Iida-nishi, Yamagata 990-9585, Japan [2] 2Internal Medicine, Yamagata
University School of Medicine, 2-2-2 Iida-nishi, Yamagata 990-9585, Japan.
  British journal of cancer (England)   May 17 2004,  90  (10)  p2013-6,
ISSN 0007-0920   Journal Code: 0370635
  Document type: Journal Article
  Languages: ENGLISH
  Main Citation Owner: NLM
  Record type: In Data Review
```

While chromosomal instability is a common feature of human solid tumours, no abnormalities in genes involved in the **mitotic checkpoint** have been identified. However, recently, **Chfr** (checkpoint with **forkhead** associated and **ring finger**), a **mitotic** stress **checkpoint** gene, has been reported to be inactivated due to promoter hypermethylation in several types of human malignancy. To clarify whether **Chfr** promoter hypermethylation is involved in gastric carcinogenesis, we investigated the promoter methylation status of the **Chfr** gene in gastric cancer cell lines and primary gastric cancers. Non-neoplastic gastric epithelia from cancer-bearing and noncancer-bearing stomachs were also examined for **Chfr** promoter hypermethylation to study its cancer specificity. Two of 10 gastric cancer cell lines (20%) showed **Chfr** promoter hypermethylation with resultant loss of expression, which could be restored by 5-aza-2' deoxycytidine treatment. **Chfr** promoter hypermethylation was present in 35% (25 of 71) of primary tumours and occurred at similar frequencies in early and advanced stages. As for non-neoplastic gastric epithelia, 1% (one of 91) from noncancer-bearing and 5% (four of 71) from cancer-bearing stomachs exhibited **Chfr** promoter hypermethylation. Thus, **Chfr** promoter hypermethylation is mostly cancer specific and frequently leads to chromosome instability in gastric cancer.British Journal of Cancer (2004) 90, 2013-2016. doi:10.1038/sj.bjc.6601849 www.bjcancer.com Published online 27 April 2004

Promoter hypermethylation of the **Chfr** gene in neoplastic and non-neoplastic gastric epithelia.

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... of 91) from noncancer-bearing and 5% (four of 71) from cancer-bearing stomachs exhibited **Chfr** promoter hypermethylation. Thus, **Chfr** promoter hypermethylation is mostly cancer specific and frequently leads to chromosome instability in gastric cancer...

6/3,K,AB/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

15564635 PMID: 14694445
CHFR -associated early G2/M checkpoint defects in breast cancer cells.
Erson Ayse E; Petty Elizabeth M
Department of Human Genetics, University of Michigan, Ann Arbor, Michigan 48109-0638, USA.
Molecular carcinogenesis (United States) Jan 2004, 39 (1) p26-33, ISSN 0899-1987 Journal Code: 8811105
Contract/Grant No.: R01 CA72877-05; CA; NCI
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM

Record type: Completed
Cell division is a highly regulated process. Checkpoints can halt cell-cycle progression due to adverse conditions such as misalignment of chromosomes to prevent missegregation. The search for new regulators of the cell cycle revealed the **mitotic checkpoint** gene **CHFR** (**checkpoint** with **forkhead**-associated and **ring finger**). **CHFR** coordinates an early mitotic phase by delaying chromosome condensation in response to a mitotic stress. Because aneuploidy and chromosome instability are common in malignant breast tumors, we screened 24 breast cancer cell lines for **CHFR** expression and demonstrated that 50% (12 of 24) of breast cancer cell lines had low **CHFR** levels. Expression of **CHFR** was reactivated with the demethylating agent 5-aza-2'-deoxycytidine (5-aza-dC) in two low-**CHFR**-expressing cell lines. Eleven of these 12 (92%) low-**CHFR**-expressing cell lines had an unusually high number of condensed chromosomes and high mitotic indices in response to nocodazole treatment. Transfection of **CHFR** in one of these cancer cell lines lowered the mitotic index after nocodazole treatment. In conclusion, our data suggested that low **CHFR** expression associated with high mitotic indices in response to nocodazole treatment were common in the breast cancer cell lines studied. Additional flow cytometry studies and analysis of a protein that interacts with **CHFR** in vitro, polo-like kinase 1 (PLK1), suggests that this **CHFR** -associated early G(2)/M checkpoint is complex, involving additional, as yet unidentified, proteins. Further analysis of **CHFR** in breast cancer cells will be important for understanding the complex mechanisms leading to aneuploidy and chromosomal instability observed in breast cancer. Copyright 2003 Wiley-Liss, Inc.

CHFR -associated early G2/M checkpoint defects in breast cancer cells.
... chromosomes to prevent missegregation. The search for new regulators of the cell cycle revealed the **mitotic checkpoint** gene **CHFR** (**checkpoint** with **forkhead**-associated and **ring finger**). **CHFR** coordinates an early mitotic phase by delaying chromosome condensation in response to a mitotic stress...

...instability are common in malignant breast tumors, we screened 24 breast cancer cell lines for **CHFR** expression and demonstrated that 50% (12 of 24) of breast cancer cell lines had low **CHFR** levels. Expression of **CHFR** was reactivated with the demethylating agent 5-aza-2'-deoxycytidine (5-aza-dC) in two low-**CHFR**-expressing cell lines. Eleven of these 12 (92%) low-**CHFR**-expressing cell lines had an unusually high number of condensed chromosomes and high mitotic indices in response to nocodazole treatment. Transfection of **CHFR** in one of these cancer cell lines lowered the mitotic index after nocodazole treatment. In conclusion, our data suggested that low **CHFR** expression associated with high mitotic indices in response to nocodazole treatment were common in the...

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Chemical Name: **CHFR** protein, human; Cell Cycle Proteins; Enzyme Inhibitors; Neoplasm Proteins; 5-aza-2'-deoxycytidine; Azacitidine

6/3,K,AB/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12422760 PMID: 12810945

Epigenetic inactivation of **CHFR** in human tumors.

Toyota Minoru; Sasaki Yasushi; Satoh Ayumi; Ogi Kazuhiro; Kikuchi Takefumi; Suzuki Hiromu; Mita Hiroaki; Tanaka Nobuyuki; Itoh Fumio; Issa Jean-Pierre J; Jair Kam-Wing; Schuebel Kornel E; Imai Kohzoh; Tokino Takashi

Department of Molecular Biology, Cancer Research Institute, Sapporo Medical University, Sapporo 060-8556, Japan.

Proceedings of the National Academy of Sciences of the United States of America (United States) Jun 24 2003, 100 (13) p7818-23, ISSN 0027-8424 Journal Code: 7505876

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Cell-cycle checkpoints controlling the orderly progression through mitosis are frequently disrupted in human cancers. One such checkpoint, entry into metaphase, is regulated by the **CHFR** gene encoding a protein possessing **forkhead**-associated and **RING finger** domains as well as ubiquitin-ligase activity. Although defects in this checkpoint have been described, the molecular basis and prevalence of **CHFR** inactivation in human tumors are still not fully understood. To address this question, we analyzed the pattern of **CHFR** expression in a number of human cancer cell lines and primary tumors. We found CpG methylation-dependent silencing of **CHFR** expression in 45% of cancer cell lines, 40% of primary colorectal cancers, 53% of colorectal adenomas, and 30% of primary head and neck cancers. Expression of **CHFR** was precisely correlated with both CpG methylation and deacetylation of histones H3 and H4 in the CpG-rich regulatory region. Moreover, CpG methylation and thus silencing of **CHFR** depended on the activities of two DNA methyltransferases, DNMT1 and DNMT3b, as their genetic inactivation restored **CHFR** expression. Finally, cells with **CHFR** methylation had an intrinsically high mitotic index when treated with microtubule inhibitor. This means that cells in which **CHFR** was epigenetically inactivated constitute loss-of-function alleles for **mitotic checkpoint** control. Taken together, these findings shed light on a pathway by which **mitotic checkpoint** is bypassed in cancer cells and suggest that inactivation of checkpoint genes is much more widespread than previously suspected.

Epigenetic inactivation of **CHFR** in human tumors.

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Chemical Name: Cell Cycle Proteins; **Chfr** protein; Chromatin; Histones; DNMT1 protein; DNMT3b; DNA (Cytosine-5-)-Methyltransferase;

Histone Deacetylases
?

```
? ds

Set      Items   Description
S1        1921   (MITOTIC(5N)CHECKPOINT) OR CHFR
S2        2842   FORKHEAD
S3          12   S1 AND S2
S4        5697   RING(5N)FINGER
S5           7   S3 AND S4
S6           3   RD (unique items)
? s s2 and s4
            2842  S2
            5697  S4
      S7      16  S2 AND S4
? rd
>>>Duplicate detection is not supported for File 340.

>>>Records from unsupported files will be retained in the RD set.
...completed examining records
      S8        9  RD (unique items)
? s s8 not s6
               9  S8
               3  S6
      S9       6  S8 NOT S6
? t s9/3,k,ab/1-6

 9/3,K,AB/1     (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2004 The Dialog Corp. All rts. reserv.

15494363   PMID: 12947006
  Insights into the multistep transformation of MGUS to myeloma using
microarray expression analysis.
  Davies Faith E; Dring Ann M; Li Cheng; Rawstron Andrew C; Shammas Masood
A; O'Connor Sheila M; Fenton James A L; Hideshima Teru; Chauhan Dharminder;
Tai Isabella T; Robinson Elizabeth; Auclair Daniel; Rees Karen; Gonzalez
David; Ashcroft A John; Dasgupta Ranjit; Mitsiades Constantine; Mitsiades
Nicholas; Chen Lan B; Wong Wing H; Munshi Nikhil C; Morgan Gareth J;
Anderson Kenneth C
  Academic Unit of Haematology and Oncology, Algernon Firth Bldg, School of
Medicine, University of Leeds, Leeds, United Kingdom. faith@egu.leeds.ac.uk
  Blood (United States) Dec 15 2003, 102 (13) p4504-11, ISSN
0006-4971 Journal Code: 7603509
  Contract/Grant No.: 50947; PHS; CA78373; CA; NCI; CA96470; CA; NCI;
HG02341; HG; NHGRI
  Document type: Journal Article
  Languages: ENGLISH
  Main Citation Owner: NLM
  Record type: Completed
  To define specific pathways important in the multistep transformation
process of normal plasma cells (PCs) to monoclonal gammopathy of uncertain
significance (MGUS) and multiple myeloma (MM), we have applied microarray
analysis to PCs from 5 healthy donors (N), 7 patients with MGUS, and 24
patients with newly diagnosed MM. Unsupervised hierarchical clustering
using 125 genes with a large variation across all samples defined 2 groups:
N and MGUS/MM. Supervised analysis identified 263 genes differentially
expressed between N and MGUS and 380 genes differentially expressed between
N and MM, 197 of which were also differentially regulated between N and
MGUS. Only 74 genes were differentially expressed between MGUS and MM
samples, indicating that the differences between MGUS and MM are smaller
than those between N and MM or N and MGUS. Differentially expressed genes
included oncogenes/tumor-suppressor genes (LAF4, RB1, and disabled homolog
2), cell-signaling genes (RAS family members, B-cell signaling and
NF-kappaB genes), DNA-binding and transcription-factor genes (XBP1, zinc
finger proteins, forkhead box, and ring finger
```

proteins), and developmental genes (WNT and SHH pathways). Understanding the molecular pathogenesis of MM by gene expression profiling has demonstrated sequential genetic changes from N to malignant PCs and highlighted important pathways involved in the transformation of MGUS to MM.

... B-cell signaling and NF-kappaB genes), DNA-binding and transcription-factor genes (XBP1, zinc **finger** proteins, **forkhead** box, and **ring finger** proteins), and developmental genes (WNT and SHH pathways). Understanding the molecular pathogenesis of MM by...

9/3,K,AB/2 (Item 1 from file: 55)
DIALOG(R)File 55:Biosis Previews(R)

0013558381 BIOSIS NO.: 200200151892
Novel transcription factors in CD34+ cells
AUTHOR: Sharma Tiffany T (Reprint); Gomes Ignatius (Reprint); Edassery Seby (Reprint); Mar Brenton (Reprint); Westbrook Carol A (Reprint)
AUTHOR ADDRESS: Dept of Medicine, Section of Hem/Onc, University of Illinois at Chicago, Chicago, IL, USA**USA
JOURNAL: Blood 98 (11 Part 2): p130b November 16, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: 43rd Annual Meeting of the American Society of Hematology, Part 2 Orlando, Florida, USA December 07-11, 2001; 20011207
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Transcription factors (TFs) and the regulatory proteins that control them play key roles in hematopoiesis, controlling the basic processes of cell growth and differentiation. Disruption of these processes may lead to leukemogenesis, and thus it is not surprising that most leukemic translocations alter genes in this category, such as AML1, PML1, and MYC. In this study, we attempted to identify novel TFs or their regulatory proteins that are expressed in undifferentiated hematopoietic tissue. We surveyed our previously-described database of 15,970 genes/ESTs representing the transcriptosome of normal human CD34+ cells by text descriptor of the UniGene annotation, and by homology with a murine stem cell database (SCdb) (http://stemcell.princeton.edu/). First, the UniGene annotation of the CD34+ database was searched for the following terms: nuclear factor, transcription factor, leucine zipper, **ring finger**, zinc **finger**, helix-loop-helix, PHD, POU, **forkhead**, bromodomain, homeobox, oncogene, nuclear, (co) activator, and (co) repressor. A total of 286 unique genes were found. Then, the human homologs of 161 genes comprising the "trancription factor" and "chromatin protein" subsets of the SCdb were identified by cross-reference to the human UniGene database, resulting in 115 genes. 73 of 115 genes were present in the CD34+ database and of these, 49 had not been identified by the previous search. These 49 genes were then verified by BLASTN sequence alignment for their gene assignment by UniGene algorithm, resulting in 36 genes with E-value less than e-40. Combining genes from both search methods produced 322 genes. Each gene was then exhaustively searched in the literature to determine if it was well-known (W), partially characterized (P) or novel, no functional study (N); chromosomal location was indicated when possible. Of the 322 genes, at least 130 were likely transcription factors, including 44 N or P. Among the novel genes identified are a p38-interacting protein (P38IP), a cDNA similar to BTF3, a transcriptional regulator protein (HCNGP), and a leucine zipper transcription factor-like protein (LZTFL1). An additional

152 N or P potential transcriptional regulators were identified in categories other than transcription factor. Thus, we describe a total of 322 TFs and putative transcriptional regulators that are likely to be expressed in CD34+ cells, and may play a role in normal and malignant growth of this tissue. The identification of these novel genes will add a wealth of information to our understanding of the molecular aspect of hematopoiesis and hematopoietic disorders.

...ABSTRACT: the CD34+ database was searched for the following terms: nuclear factor, transcription factor, leucine zipper, **ring finger**, zinc **finger**, helix-loop-helix, PHD, POU, **forkhead**, bromodomain, homeobox, oncogene, nuclear, (co) activator, and (co) repressor. A total of 286 unique genes...

9/3,K,AB/3 (Item 2 from file: 55)
DIALOG(R)File 55:Biosis Previews(R)

0011373789 BIOSIS NO.: 199800168036
The pufferfish SLP-1 gene, a new member of the SCL/TAL-1 family of transcription factors
AUTHOR: Gottgens Berthold; Gilbert James G R; Barton Linda M; Aparicio Samuel; Hawker Kelvin; Mistry Shailesh; Vaudin Mark; King Andrew; Bentley David; Elgar Greg; Green Anthony R (Reprint)
AUTHOR ADDRESS: Dep. Haematol., MRC Centre, Univ. Cambridge, Hills Rd., Cambridge CB2 2QH, UK**UK
JOURNAL: Genomics 48 (1): p52-62 Feb. 15, 1998 1998
MEDIUM: print
ISSN: 0888-7543
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The SCL/TAL-1 gene encodes a basic helix-loop-helix (bHLH) transcription factor essential for the development of all hemopoietic lineages and also acts as a T-cell oncogene. Four related genes have been described in mammals (LYL-1, TAL-2, NSCL1, and NSCL2), all of which exhibit a high degree of sequence similarity to SCL/TAL-1 in the bHLH domain and two of which (LYL-1 and TAL-2) have also been implicated in the pathogenesis of T-cell acute lymphoblastic leukemia. In this study we describe the identification and characterization of a pufferfish gene termed SLP-1, which represents a new member of this gene family. The genomic structure and sequence of SLP-1 suggests that it forms a subfamily with SCL/TAL-1 and LYL-1 and is most closely related to SCL/TAL-1. However, unlike SCL/TAL-1, SLP-1 is widely expressed. Sequence analysis of a whole cosmid containing SLP-1 shows that SLP-1 is flanked upstream by a zinc finger gene and a **forkhead**-domain gene and downstream by a heme-oxygenase and a **RING finger** gene.

...ABSTRACT: 1 shows that SLP-1 is flanked upstream by a zinc finger gene and a **forkhead**-domain gene and downstream by a heme-oxygenase and a **RING finger** gene.

9/3,K,AB/4 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

12465335 Genuine Article#: 768ZF Number of References: 126
Title: Nuclear transport and cancer: From mechanism to intervention (
ABSTRACT AVAILABLE)

Author(s): Kau TR; Way JC; Silver PA (REPRINT)
Corporate Source: Harvard Univ,Sch Med, Dept Biol Chem & Mol Pharmacol,Boston//MA/02115 (REPRINT); Harvard Univ,Sch Med, Dept Biol Chem & Mol Pharmacol,Boston//MA/02115; Dana Farber Canc Inst,Dept Canc Biol,Boston//MA/02115; EMD Lexigen Res Ctr,Billerica//MA/01821
Journal: NATURE REVIEWS CANCER, 2004, V4, N2 (FEB), P106-117
ISSN: 1474-175X Publication date: 20040200
Publisher: NATURE PUBLISHING GROUP, MACMILLAN BUILDING, 4 CRINAN ST, LONDON N1 9XW, ENGLAND
Language: English Document Type: REVIEW
Abstract: Nuclear-cytoplasmic transport, which occurs through special structures called nuclear pores, is an important aspect of normal cell function, and defects in this process have been detected in many different types of cancer cells. These defects can occur in the signal-transduction pathways that regulate the transfer of factors such as p53 and beta-catenin in and out of the nucleus, or in the general nuclear import and export machinery itself. In some cases, nuclear transport factors are overproduced, whereas in others, chromosomal translocations disrupt the structural proteins that make up the nuclear pore, leading to cell transformation. How does disruption of nuclear-cytoplasmic transport promote transformation, and is this process a viable therapeutic target?

...Identifiers--**FORKHEAD** TRANSCRIPTION FACTOR; NF-KAPPA-B; PROTEIN-KINASE-B; TUMOR-SUPPRESSOR PROTEIN; SEGREGATION GENE CSE1; WILD-TYPE P53; APOPTOSIS SUSCEPTIBILITY GENE; **RING-FINGER** DOMAIN; CELL-CYCLE ARREST; BREAST-CANCER

9/3,K,AB/5 (Item 2 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

11307076 Genuine Article#: 636BF Number of References: 74
Title: Nuclear transport as a target for cell growth (ABSTRACT AVAILABLE)
Author(s): Kau TR (REPRINT) ; Silver PA
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Abstract: The function of many key proteins and transcription factors involved in cell growth can be regulated by their cellular localization. Such proteins include the tumor suppressor p53 and the nuclear factor kappaB. Although the idea of trapping such proteins in either the nucleus or cytoplasm has been introduced as a potential therapeutic target, only two nuclear transport inhibitors have been reported. Here, we explore the roles of small-molecule inhibitors that cause target proteins to sequester in either the nucleus or cytoplasm. Methods of artificially targeting proteins to the nucleus or cytoplasm using peptide aptamer technology are also discussed.

...Identifiers--NF-KAPPA-B; PROTEIN-KINASE-B; **FORKHEAD** TRANSCRIPTION FACTOR; ORDER CHROMOSOME STRUCTURE; TUMOR-SUPPRESSOR PROTEIN; **RING-FINGER** DOMAIN; LEPTOMYCIN-B; CAENORHABDITIS-ELEGANS; PEPTIDE APTAMERS; EXPORT SIGNAL

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Abstract: A method of identifying a polynucleotide or pattern of polynucleotides regulated by one or more sepsis or inflammatory inducing agents and inhibited by a peptide is described. A method of identifying a pattern of polynucleotide expression for inhibition of an inflammatory or septic response. The method includes contacting cells with LPS, LTA, CpG DNA and/or intact microbe or microbial components in the presence or absence of a cationic peptide; detecting a pattern of polynucleotide expression for the cells in the presence and absence of the peptide, wherein the pattern in the presence of the peptide represents inhibition of an inflammatory or septic response. Also included are compounds and agents identified by the methods of the invention. In another aspect, the invention provides methods and compounds for enhancing innate immunity in a subject.

Non-exemplary Claims: ...31. The method of claim 30, wherein the pro-inflammatory polynucleotides include **ring finger** protein 10 (D87451), serine/ threonine protein kinase MASK (AB040057), KIAA0912 protein (AB020719), KIAA0239 protein (D87076...

...protein (AF061261); Cell cycle gene (S70622); IL-10 Receptor U00672); Transferase (AF038664); Homeobox protein (AC004774); **Forkhead** protein (AF042832); Unknown (AL096803); KIAA0284 Protein (AB006622); Hypothetical Protein (AL022393); Receptor (AF112461); Hypothetical Protein (AK002104...

...state of infection identified by claim 56 wherein the genes upregulated are Accession number D87451 -**ring finger** protein 10; Accession number AL049975, Unknown; Accession number U39067, eukaryotic translation initiation factor 3 subunit...
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